SCARLATINAL NEPHRITIS.[1]

By I. E. Atkinson, M.D.,
PROFESSOR OF THERAPEUTICS AND MATERIA MEDICA AND CLINICAL MEDICINE IN THE UNIVERSITY OF MARYLAND.

Scarlet fever derives most of its dangers from disorders that complicate or succeed it. Of these, the most important are derangements of the kidneys. Indeed, a number of recent writers assert that these organs are constantly affected in scarlet fever. Eisenschitz declares that renal catarrh is just as much a feature of scarlet fever as bronchial catarrh is of measles. Frerichs, Reinhardt, Begbie, Newbigging, Holder, and others, maintain the same opinion. The three last-mentioned authors found albuminuria in their cases of scarlet fever without exception (Gee). Böning, who likewise regards renal derangement as constant, found it only to a slight degree in most of his cases, but often, also, encountered diffuse croupous nephritis, which, however, only seldom passed into chronic parenchymatous nephritis.[2]

Future research may show that simple renal catarrh accompanies all cases of scarlatina, but it is certainly not true that renal alterations competent to excite albuminuria or to reveal themselves *post mortem* to reasonably careful inspection, are invariably present. Thomas practised microscopic examinations in twenty-five of eighty patients, and in twenty of these daily. In the prodromal and eruptive stages he found albuminuria only rarely, and then it was transitory and very slight. Signs of a decided alteration in the urinary tract were most uncommon—symptoms of mild catarrh were more often seen. Only the more severe forms were considered by Thomas to depend upon a specific scarlatinal influence.[3] Pathological anatomy would seem to bear out such conclusions, for although Steiner states that evidences of kidney disorder are always present in those who die of scarlatina, whether signs of it were present during life or not, Friedländer, who examined the bodies of 229 scarlet fever cases, after death, found kidney disorder in a little less than one-half. Moreover, it must be admitted that fatal cases of scarlatina are vastly more apt to present renal disorder than those which recover.

Renal catarrh, however, is much more frequently an accompaniment of scarlatina than is generally supposed. It usually escapes observation, as it is only exceptionally revealed by symptoms and can only be recognized after microscopic examination of the urine, a procedure too often neglected, but of the greatest importance as often anticipating dangerous processes that may be averted by timely treatment. Thomas, in denying that this condition is at all constant, shows that it also occurs in measles, croupous pneumonia, intestinal catarrh, etc., and is often but an expression of the febrile condition. At the same time, the catarrh is relatively so frequent in scarlatina that he cannot avoid the conclusion that the specific influence of the disease is often concerned in its production, especially as there is frequently a disproportion between the degree of fever and the signs of renal irritation. Careful observation will detect in many cases from the very beginning, heaped-up masses of renal epithelium, like cylinders, some more or less degenerated or changed into detritus. In milder cases the urine will contain long, flattened mucous casts with increased quantity of mucus, but no albumen. In severe cases the urinary sediment will show more hyaline casts studded with epithelium and epithelial débris, and red and white blood-corpuscles. Slight albuminuria will also be present. This catarrh is for the most part insignificant, and probably only rarely serves as the starting-point for the graver, pronounced, and characteristic forms of nephritis scarlatinosa, though, without doubt, many milder forms of scarlatinal nephritis and dropsy originate in it. Böning and others have observed it pass into croupous parenchymatous nephritis. Thomas concluded that those cases in which nephritis develops suddenly and not from preceding renal catarrh, usually end fatally.

---

[1] Read before the Clinical Society of Maryland.

[2] In a paper read before the Royal Medical and Chirurgical Society, a synopsis of which appeared in the *Lancet* (Nov. 14, 1885), Dr. Stevenson Thomson, after examination of the urine of 180 cases of scarlet fever, favored the view that nephritis is a feature of scarlet fever almost as essential as the rash or sore throat.

[3] Jahrb. f. Kinderheilk., 1870, i.

Inflammation of the kidneys sufficiently pronounced to excite albuminuria varies greatly in the relative frequency of its occurrence. In some epidemics it appears in as many as 70 per cent. of cases, while in others not more than 5 per cent. may be attacked. Dickinson, in quoting Hillier's observation that at the Children's Hospital about half of the cases of scarlatina had albuminuria, considered the statement as rather below than above the truth.[1] Fleischmann, at St. Joseph's Hospital in Vienna, noted 95 cases of Bright's disease in 472 observations. During 1861, every third child with scarlatina had dropsy; while during 1862, it developed in only one in ten.[2] Thomas asserts that renal alterations develop in about one-half of all cases of scarlet fever; while, as has been already shown, there are those who assert that they are present in every case. On the other hand, Jaccoud declares that for fifteen years he has never had a case of scarlatinal nephritis among his scarlet fever patients, a result that he attributes to his treatment.[3] Albuminuria may appear at any time during the attack of scarlatina from the very first day, though its most common occurrence is during the second and third weeks. Obviously, dropsy should not be taken as marking the beginning of nephritis, the signs of which may be present in the urine for hours, even days, before this occurs. Fleischmann noted the appearance of dropsy in his cases, 9 times during the first week, 30 times during the second week, 23 times during the third week, 20 times during the fourth week, and 5 times after the fourth week. Of 60 cases at the Children's Hospital, 42 began between the end of the first and the end of the fourth week, while 5 became dropsical during the first week.

---

[1] Albuminuria, page 320.

[2] Jahrb. f. Kinderheilk., 1870, 411.

[3] Gazette des Hôpitaux, 58, 1885, 418.

When nephritis occurs during the first week of scarlatina, it often escapes attention at the beginning, on account of the blending of its symptoms with those of the essential disease and of the neglect of the attendant to examine the urine. Dropsy will, of course, attract notice, but this does not often occur so early, and may be confounded with the œdema due to the exanthem. Rarely, the fatal issue of what was, apparently, malignant scarlet fever, really may have resulted from a uræmic poisoning due to a fulminating nephritis. In either case the symptoms may have been identical. Fever, vomiting, headache, delirium, amblyopia, coma, convulsions, may have been present. The convulsions are often very irregular. They may be general, partial or unilateral, tonic or clonic. The patient may have several in rapid succession, or may pass into a status epilepticus from which only death will release him. In nephritis of this character the urine will be completely or partially suppressed. That secreted will be of high specific gravity (1.020 to 1.040), dark and smoky in appearance, loaded with albumen, and depositing an abundant sediment of hyaline, epithelial, granular, and blood-tube-casts, with renal epithelium and red and white blood-corpuscles in greater or less quantity.

According to Glax[1] and others, the very diminution, to great extent, of the urine, during scarlatina, is ominous, as indicating renal complications, and death has been known to follow before albuminuria was established. If the kidneys become implicated toward the end of the first week, the development of symptoms may delay the course of what may otherwise appear to be a normal case of scarlet fever. Microscopical research will often betray the onset of the changes in advance of chemical analysis; casts of the renal tubules will be observed, along with epithelial deposits and detritus, before albuminuria is developed. This, however, will shortly appear, and in severe cases the scarlatinous symptoms will be interrupted by those of the nephritis. There will be no constant relation between the amount of albumen, the tube-casts, and the general detritus—one variety of sedimentary matter being at one time copious, at another very scanty. At this time vomiting may begin with returning headache, the appetite may again fail, pain in the loins may become annoying; the patient may again become depressed and feeble, and his fever may cease to diminish—may even exceed its original intensity. At other times no apparent influence will be exerted upon the scarlatina, which will run its usual favorable course until the occurrence of dropsy and albuminuria reveals the implication of the kidneys.

---

[1] Deutsche Archiv f. klin. Med., 33, 1883.

When the renal disorder develops after defervescence, during the second, third, or fourth week, or later, the same series of symptoms may be observed, their severity being generally in direct ratio to the earliness of their occurrence. Thomas thinks that cases developing after the fourth week may be expected to run a favorable course. It has been asserted that the renal disorder may arise after several months; but if a patient pass the sixth week in safety, he will almost certainly escape. The symptoms, in cases arising during these weeks are not always gradually developed, and some of the most disastrous results of the disease may be encountered during the second, third, or fourth weeks in children apparently convalescing from scarlatina, and often in full desquamation, who, after a few hours' indisposition with nausea, headache, malaise, confusion of ideas, or stupor, with return of fever, rapidly pass into coma or convulsions ending after a short interval in death, before dropsy has developed and after partial or complete suppression of urine.

It is the rule for scarlatinal nephritis to pursue a mild and favorable course. Dropsy is usually the first symptom observed, first appearing in the face, and sometimes remaining confined to that locality; at other times becoming speedily general, and giving an appearance of plumpness, but with a waxy translucency of skin. The face, upper and lower extremities, body wall, and prepuce may thus become dropsical. The serous cavities are very often implicated, and more or less filling of the pericardial, pleural,[1] peritoneal, scrotal, and intracranial cavities occurs. Œdema of the lungs and of the glottis not very infrequently imperils life. Desquamation is often completely arrested upon the supervention of dropsy. The temperature, which in the more severe cases may be much elevated, is more commonly but little above normal, not exceeding 101° or 102° F. The pulse, sometimes accelerated and feeble, will often become remarkably slow and intermittent, and so remain throughout the attack. The child will grow dull and listless, and exceedingly feeble. Pain in the belly and in the back will at times prove very annoying, or, again, may be absent. The tongue, having lost the strawberry aspect of the stage of eruption, will become pale, flabby, and coated, the appetite will fail, and the bowels become sluggish. The urine will rapidly diminish in quantity, and may deposit urates copiously, or may present a smoky and oily appearance, due to the abundant presence of epithelial cells, white and red blood-corpuscles, and tube-casts. The total amount may now be reduced to a few ounces. Blood corpuscles are often very abundant, and form a thick red layer at the bottom of the test-tube. This free admixture of blood may at times amount to very well-marked hæmaturia. Such hæmaturia is generally post-scarlatinal, and, according to Schütz, occurs during the third or fourth week most frequently. Ordinarily, of itself, it adds but little to the gravity of the case. The patient often feels fairly well, and may eat and sleep with comfort. While the œdema and pallor may be very great, the temperature and pulse may vary but little from the normal, or may show the changes of ordinary scarlatinal nephritis. With the gradual improvement of all the symptoms, the hæmaturia disappears. Heubner[1] has reported a case of nephritis after scarlatina in which hæmoglobinuria was present. The urine became brownish-black; no blood corpuscles were present. Death resulted from asthenia on the fifth day after both albumen and hæmoglobin had disappeared from the urine.

---

[1] The hydrothorax is sometimes unilateral.

The amount of albumen in the urine in scarlatinal nephritis is usually very great. The urinary sediment is also abundant, and is largely composed of tube-casts. Hyaline casts predominate; though finely and coarsely granular, and epithelial casts are numerous; later, coarse fatty granules stud the casts plentifully. The casts are often almost diffluent, and differ strikingly from the firm and sharply outlined casts of more chronic nephritis. They are sometimes of enormous size, coming from the larger tubules. Crystalline deposits are scanty, and are mostly confined to uric acid and urates. On the other hand, the amorphous urates are often very abundant. The extent of the albuminuria present is of less importance than the total quantity of urine, rapid and extensive diminution of this being most ominous as indicating the accumulation of nitrogenous waste in the blood and danger of resulting uræmia. Glax[2] has been able to draw important conclusions from the amount of urinary water in scarlatina. He recognizes three types, after which the urine is secreted: 1. The total urine is only lessened so long as the fever continues, after which it gradually increases until the normal amount is reached, or, for a few days, even exceeded. In such cases the course is always favorable, desquamation occurs promptly, and albuminuria is absent. 2. Diuresis increases after the beginning of fever, often very considerably, then during the following days markedly diminishes, and shows notable variations during the attack. In these cases the course is protracted, desquamation imperfect, and the heart's action weak. Anæmia frequently develops, cutaneous œdema not seldom, but the urine remains free from albumen. 3. Micturition, which during the fever was much diminished, quickly returns to the normal with defervescence, and then suddenly again diminishes, and remains scanty until death, or after several days polyuria develops, which slowly sinks to normal diuresis. These are the cases in which, after a normal course, nephritis and dropsy occur later. A lessening of the proportion of urine secreted to fluid ingested (2 : 3) not unfrequently foreshadows the approach of uræmic symptoms, even though the urine contains no albumen.

---

[1] Deutsches Arch. f. klin. Med., xxiii. 288.

[2] Ibid., xxxiii., 1883.

Whether, throughout the attack, the temperature remain normal or after an initial chill become elevated and all the symptoms of acute nephritis develop, complete recovery may reasonably be expected if the patient pass safely through the earlier phases of the disorder. But although nephritis and dropsy may be slight, albuminuria lasting only a few days and anasarca being limited to mild puffiness about the eyes, scarlatinal nephritis does not usually subside entirely in less than a month. It may endure as long as three, four, or five months, and, rarely, there seems good reason to believe that chronic nephritis in young people may have had its beginning in antecedent scarlatinal inflammation of the kidneys. Such a result, however, is exceedingly uncommon.

The dropsy is indicative of the degree of renal derangement, except in the most acute cases, and sometimes reaches enormous proportions—as the urine increases in quantity the albumen proportionally diminishes and the dropsy disappears; the skin, which until now has been dry and inactive, becomes softer, more elastic, and resumes its proper functions. The appetite improves, the spirits, strength, and mental activity return, and good health gradually becomes restored. Just as the microscope reveals the earliest evidence of renal derangement, so does it continue to expose the results of pathological action after chemical tests fail to do so. Tube-casts continue to appear in the urinary sediment, sometimes for weeks after the cessation of albuminuria, the epithelial, blood, coarsely granular, and fatty casts gradually giving place to finely granular, hyaline, and mucous ones, which, in their turn, finally disappear. When the disorder terminates fatally, the symptoms will be those of acute nephritis: suppression of urine may be followed by signs of cerebral disturbance, headache of violent character, during which blindness occurs very often, with or without dilatation of the pupil, vomiting and convulsions, partial or general, coma, and sometimes paralysis; or the fatal termination may slowly be reached through constantly increasing asthenia; or, what is quite frequent, complications may arise which cannot always definitely be ascribed to the nephritis or to the scarlatina itself. These are inflammations of the pleuræ, the pericardium, the endocardium, the peritoneum, the cerebral meninges, etc. Pneumonia, acute articular rheumatism, or enteritis, may also hasten the fatal issue.

Cases are occasionally observed in which dropsy follows scarlatina, but without albuminuria. Indeed, a tendency toward non-albuminuric dropsy after scarlatina has been associated with certain epidemics. Scarlatinal dropsy without renal affection has been observed by Guersant, Rilliet and Barthez, Noirot, Bouchut, Löschner, Duckworth, and others. Quincke[1] tries to explain such cases of non-albuminuric dropsy as not depending upon nephritis, but as a consequence of the scarlatinous irritation exerting some peculiar influence upon the connective tissue. Such cases probably occur in the experience of most busy practitioners. One should be cautious, however, in deciding against a nephritic origin of these dropsies, except where they can be definitely attributed to anæmia and debility. Hennoch[2] has asserted that cases of nephritis occur in which albuminuria is absent up to the time of death. He reports a case in which anasarca was present for three weeks after scarlatina, and yet albuminuria and tube-casts were absent until convulsions occurred, with death resulting from œdema of the lungs. In this case, a necropsy revealed the presence of acute nephritis. He also reports the case of a child dead on the thirteenth day, of malignant scarlet fever, in whom repeated tests during life had not shown albuminuria, and yet whose kidneys showed indubitable evidences of hemorrhagic nephritis. Steiner has seen nephritis without dropsy, but never dropsy without nephritis after scarlatina. It is altogether probable, however, that in many cases the dropsy following scarlatina without albuminuria is secondary to concomitant anæmia. This is the view adopted by Hennoch. Whatever be the explanation, such cases usually run no remarkable course. The general health is not much reduced. The urine is in normal amount, the various functions fairly performed. With the disappearance of the dropsy, convalescence generally becomes established.

Scarlatinal nephritis is not associated with any especial type or phase of scarlatina. It is as frequent after mild as after severe attacks; indeed, it is possible that the care exercised over those who have had grave attacks of scarlet fever, as shown in proper nursing and hygienic surroundings, may furnish a safeguard against renal complications. At all events, there is a widespread belief that the milder cases are more apt to be followed by nephritis and dropsy. Violent nephritis may certainly follow a scarlatina so mild as to have escaped observation; but individual predisposition and epidemic type are probably the most important etiological factors, though at present not enough is known to justify dogmatic statement.

Scarlatinal nephritis and dropsy may occur without antecedent symptoms of scarlatina. Instances of this are not uncommon, though the evidence that supports them is rather circumstantial than direct. Several members of a family, or of a school or asylum in which scarlatina has been known to prevail, may exhibit the dropsy and albuminuria characteristic of scarlet fever, in whom no previous symptom of the disease had been manifested. Such cases run an ordinary course, but may at times develop a severity altogether unexpected.

---

[1] Berl. klin. Wochensch., 27, 1882.

[2] Ibid., 50, 1873.

PATHOLOGICAL ANATOMY.—The kidneys of those who die of scarlatina, or of the nephritis consequent upon it, present morbid conditions, the often widely differing characters of which depend upon the nature and intensity of their exciting causes. Friedländer,[1] in his investigations, found three forms of renal inflammation after scarlatina, which are sharply defined from each other. These are, 1. Initial catarrhal nephritis, the early form; 2. The big, flabby, hemorrhagic kidney, interstitial septic nephritis; 3. Glomerulo-nephritis, nephritis post-scarlatinosa. The first, he asserts, appears with the beginning of the exanthema, or a few days later, and disappears in a few days or weeks. It rarely excites œdema, and hardly ever kills. It is analogous to the alterations productive of the febrile albuminuria of many infectious diseases. The kidneys show cloudiness, swelling and proliferation of the tubular epithelium, and later, fatty degeneration. In the tubular lumen are hyaline and granular cylinders, round cells, and desquamated epithelium. In the interstitial tissue are scattered round cells. The capsule of Bowman is thickened, and at times there is a small amount of albuminous fluid between the capsule and the glomerulus. Micrococci are sometimes found in the capillaries and tubules. The large, flabby, hemorrhagic kidney was found in 12 of the 229 scarlatinal necropsies made by Friedländer. It was found especially when the scarlatina had been complicated by severe diphtheria, abscess, etc. It is not characteristic of scarlatina, but is also seen in primary idiopathic diphtheria. The kidneys are large and soft, and show pronounced cortical changes. The cortex is invaded by small extravasations and larger blood infiltrations. The epithelium is only slightly altered, but the interstitial tissue is thickened and abundantly infiltrated with round cells. Emboli of micrococci are commonly present. The disorder develops between the first and fourth weeks, and is so rapidly fatal that œdema has not time to develop. It is an especially severe form of septic nephritis.

Glomerulo-nephritis, Friedländer holds to be the only characteristic scarlatinal nephritis. The kidneys in this condition are firm, often hyperæmic, and resemble the cyanotic kidney, except that the glomeruli do not appear red upon section, but gray and anæmic. They are enlarged and prominent. Alterations are almost limited to the glomeruli, which are enlarged by one-half. Their nuclei are enlarged, their coils empty of blood, their walls thickened, and their lumina contracted or obliterated. Bowman's capsule is only slightly thickened, as a rule; sometimes it is proliferated. Accompanying conditions are slight interstitial cell infiltration, fatty degeneration of epithelial cells, and hyaline formation in the arteries. The alterations in the glomeruli easily account for the anuria and uræmia, as well as rapid hypertrophy of the left ventricle, by their obstruction of the renal arteries, as nearly all of the renal arterial blood has first to pass through the glomeruli. Scarlatinal glomerulo-nephritis was first described by Klebs.[1]

---

[1] Fortschritte d. Med., No. 3, 1883, p. 81.

Klein,[2] who has given the subject especial attention, in a series of 23 necropsies of bodies of those dead of scarlet fever, did not observe the identical glomerulo-nephritis as described by Klebs. Klein's cases died at various periods between 2 and 44 days. Their ages were between 2 and 36 years, the largest number being between 2 and 12 years old. Klein's observations included changes resembling the glomerulo-nephritis of Klebs, but these were only characteristic of the early stages of scarlatina. He divides the changes into those occurring in the early and those occurring in the late stages, a definite boundary between them not being present. The first set of changes are chiefly limited to the cortex. They are, 1. Increase of nuclei (probably epithelial) covering the glomeruli. 2. Hyaline degeneration of the elastic intima of minute arteries, especially of the afferent arterioles of the Malpighian tufts. The intima of these vessels is swollen from place to place into spindle-shaped hyaline masses, causing narrowing of the lumen. There is similar hyaline degeneration of the capillaries of the glomeruli, causing these often to become impermeable. These degenerated parts become more fibrous in appearance and Bowman's capsule becomes thickened. 3. A third change is multiplication of the nuclei of the muscularis of the minute arteries with increased thickness of their walls. This is most conspicuous at the entrance into glomeruli, but is also distinct in other arteries of the cortex and in the base of the pyramids. There are, also, swelling of the epithelia of the convoluted tubules and proliferation of their nuclei, especially of the tubules close to the afferent arterioles of the glomeruli. Granular matter and blood may be found in the cavity of Bowman's capsule and in the convoluted tubules. In some cases there is detachment of the epithelium of the large tubules of the pyramids.

Klein's observations, 1, that the hyaline changes readily affect the arteries near their point of branching, and, 2, that the hyaline substance is of the nature of elastic tissue, agree with the conclusions of Neilson concerning the arteries in various cerebral disorders and in many infectious diseases. He does not think that the anuria and uræmic poisoning in scarlatina, when the kidney does not show conspicuous change, are due to compression of the vessels of the glomerulus by the nuclear germination, as claimed by Klebs. He rather attributes these to the changed state of the arterioles, and suggests that the increased exercise of the arterial muscular tissue under the influence of the stimulus supplied by the disease, may cause a contractility which affects the calibre of the arterioles and shuts out the glomerulus from the circulation, and thus, so far as it operates, suppresses the secretion of urine. This hyaline change is seen in other arteries and in other diseases. The parenchymatous changes found in the early stages are slight and in some cases difficult to detect, the cloudy swelling and granular degeneration being limited to small portions of convoluted tubules. The second order of changes occurred in cases beginning about the ninth or tenth day. They were interstitial as well as parenchymatous. Round cells were found in the connective tissue of the kidneys around the large vascular trunks, spreading thence into the base of the pyramids and into the cortex. Klein declares that this begins about the end of the first week and gradually increases until portions of the cortex, very seldom portions of the bases of the pyramids, are converted into pale, firm, round cell tissue, in which the original tubes of the cortex become gradually squeezed and lost. The parenchymatous element of the nephritis consists in crowding of urinary tubules with lymphoid cells, granular and fatty degeneration of the epithelium of the tubules, and various kinds of cylinders. This becomes distinct as the interstitial changes become pronounced. The infiltration of the cortex with round cells begins at the roots of the interlobular vessels, spreading rapidly toward the capsule of the kidney, and laterally among the convoluted tubules around the glomeruli. At first between the medullary rays, it later encroaches upon them and also the subcapsular region. Parts of the cortex may thus be converted into whitish, firm, bloodless, cellular masses in which Malpighian tufts and urinary tubules become more or less degenerated. In one case renal embolism was encountered; the interstitial inflammation was very intense, as also the parenchymatous inflammation. The kidney was markedly enlarged. The intensity of the parenchymatous inflammation is dependent upon the degree of the interstitial nephritis. Klein also notes the deposition of lime in the epithelium and lumina of the tubules, first of the cortex and then of the pyramids, at an early stage of scarlatina when only slight changes are otherwise shown. Cases of scarlatina, according to Klein, that die after the ninth or tenth day, usually show more or less well-marked interstitial nephritis.

---

[1] Handbuch der Path. Anat.

[2] Transact. Path. Soc. London, 1877, xxviii. p. 435.

TREATMENT.—Though nephritis may arise both in mild and in severe cases of scarlatina, in cases where exposure is known to have occurred and in those who have been jealously protected from injurious influences, there can be no doubt that sudden changes of temperature, strong draughts, dampness, etc., are powerful predisposing causes of kidney disease after scarlatina. Mahomed declared that a slight chill during convalescence is sufficient to cause transitory albuminuria. The patient should, therefore, be carefully protected. During the height of the disease the daily bath or tepid sponging should be continued, a procedure through which renal complications are frequently avoided. Even the mildest case should be kept in bed for at least a week after the cessation of fever; nor should he be permitted to leave his room before the expiration of the third week. Out-of-door exercise should not be resumed in disregard of states of season, of barometric and of thermometric variations.

In midsummer, when windows and doors must remain open, the question of outdoor exercise becomes one of danger rather to others than of personal risk; while in spring, fall, and winter the risks of exposure are especially great, and in midwinter the patient should not venture out before the sixth or seventh week after perfectly normal scarlatina. During convalescence, daily warm or tepid baths should be continued until the completion of desquamation. Slight albuminuria may occur, according to Mahomed, during convalescence, associated with constipation and a hard pulse, indicative of high arterial tension, without subjective symptoms, and remediable by a brisk purge, the renal functions being quickly restored.

Dietary management will go far toward preventing renal complications. The observance of a rigid milk diet in all cases of scarlatina is regarded by Jaccoud as absolutely preventive of nephritis in scarlet fever. Though this may not be a justifiable opinion, it is certain that in this disease there is no better diet than one of milk. Should nephritis arise, it is the more important that the milk diet should be continued. From two to three or four pints may be given in small quantities at brief intervals, during the twenty-four hours, the largest amount mentioned being sufficient for an adult without other food. If there are reasons why milk cannot be given, light broths and soups are more suitable than beef tea and may be administered with wholesome farinaceous food. Buttermilk may at times be preferred, and bonnyclabber and slip or junket (milk sweetened and flavored and coagulated with liquid rennet), are often relished and are excellent articles of food.

When nephritis forms a feature of rapidly fatal malignant scarlatina, it may have no time to develop symptoms, or these may escape observation, or the virulence of the disease may have thrown the renal disorder into the background or have rendered attempts to treat it futile. In milder cases, and later, during the latter part of the first or during the second or third week, when nephritis occurs, special attention may be devoted to its treatment.

Proper regard having been paid to the patient's hygienic surroundings and nutrition, a brisk hydragogue cathartic should be administered, if, as is usual, there is a tendency toward constipation. For this purpose nothing is better than the compound jalap powder. For a child, from five to twenty grains of this should be ordered every second night or every night, as required, the object being to secure several watery actions of the bowels every twenty-four hours. The proper dose for an adult is one drachm. The desired watery stools will be most readily obtained by the saline cathartics when given in concentrated watery solution, as has been definitely shown by the researches of Dr. Matthew Hay. The more drastic purgatives will rarely be required except in uræmic intoxication and in extreme dropsy, when podophyllin, croton oil, elaterin, etc., may occasionally be employed with benefit. When dropsy is but slightly pronounced, purgation may not be required.

The proper action of the bowels being provided for, the establishment of diaphoresis and healthy skin action will demand attention. Frequently during the day the body may be wrapped in flannels, wet or dry, as hot as can be borne, or the pack may be applied. When available, the steam bath or hot-air bath is to be strongly recommended. Excellent results may also be secured from the hot plunge-bath. Pilz has especially lauded this treatment. It should be used after the method of Liebermeister, by gradually increasing the temperature of the bath from 36° C. to 40° C. (96°–104° F), in a half hour. Under its use the dropsy rapidly disappears. Diseases of the heart or lungs, while not positively contraindicating this plan of treatment, necessitate great caution in its application. Every precaution against sudden chilling of the surface must be observed.

The kidneys demand the greatest consideration. The imminence of danger is usually proportionate to the impairment of their function. In giving remedies directly to modify the action of the kidneys, none calculated to increase their hyperæmia should be employed. On this account, the stimulating diuretics should be avoided. Exception can hardly be made in favor of juniper, which enjoys, with some writers, considerable reputation in scarlatinal nephritis; but digitalis, the pharmacological position of which is not as yet definitely determined, has received very general approval as a most useful diuretic in acute nephritis. The infusion is by all odds the best preparation of digitalis when its diuretic properties are desired, and may be given in doses of from one fluid-drachm to a half fluidounce, according to age, three or four times daily. Its effects are, however, hardly as happy as when dropsy is associated with or dependent upon cardiac weakness. Those diuretics that act specifically upon the secreting cells of the urinary tubules, the sedative or refrigerant diuretics, are, as a rule, to be preferred in the treatment of scarlatinal dropsy, and will often effect the most astonishing results. Of these the salts of potash are most efficacious, the citrate, acetate, bitartrate, and bicarbonate. In cases of slight nephritis and anasarca, a lemonade made with bitartrate of potash will be taken with avidity, and will often almost magically increase the quantity of urine, reduce the dropsy, rapidly diminish the albuminuria, and cause a radical change for the better. This lemonade may be made by adding one drachm of cream of tartar to a pint of boiling water into which a lemon, cut into thin slices, has been dropped. This quantity, properly sweetened, may be drunk during the day by a child five years old. Water may be allowed freely, and any of the mild domestic infusions may be substituted for it, their virtue residing principally in the amount of fluid. Dickinson especially commends the free use of water as unirritating and tending to wash out of the tubules the exudated matters that choke up their lumina.

In more severe cases, where life is threatened through one or another of the various features of uræmia, most energetic treatment will be required. Jaborandi may now prove useful. J. Lewis Smith speaks of it in terms of highest praise, and quotes Hirschfeld's commendation of its action. To a child two years old one-twentieth of a grain of pilocarpin may be given by the mouth every fourth or sixth hour, or the same amount may be injected hypodermatically. Both diuresis and diaphoresis will be promptly increased, and in favorable cases the uræmic symptoms disappear. Uræmic coma and convulsions developing suddenly or only after progressive renal embarrassment, should be treated without reference to the scarlatina and upon general principles. A remedy of most undoubted value, at least for the control of convulsions, is chloral, which, if the patient be unable to swallow, may be injected in full doses under the skin or into the rectum. Under its influence convulsions will often speedily cease.

After the more acute nephritis has subsided and convalescence promises to become established, iron becomes one of our main reliances, both through its hæmatic and diuretic properties. The tincture of the chloride is the most appropriate preparation in some cases, but, in general, the mistura ferri et ammoniæ acetatis of the pharmacopœia will produce the best results. Quinine is also a remedy of great value in the treatment of convalescence from scarlatinal nephritis.

During the height of the inflammation, local treatment is often of great importance. If the fever is intense, the pulse full and strong, and if pain and tenderness in the back are pronounced, the abstraction of blood, by leeches or cups, from the loins, will, beyond doubt, often prove beneficial. Large poultices and sinapisms may also often be applied over the kidneys, and besides assuaging the irritation, tend to promote diaphoresis and diuresis. For obvious reasons turpentine should not be employed as a counter-irritant in these cases. Occasionally ascites may be so excessive that the pressure exerted upon the kidneys interferes with the action of therapeutic agents and impedes the functional activity of these organs. Paracentesis abdominis, by relieving this compression, will often be followed by copious diuresis and the rapid disappearance of general anasarca. Cases of scarlatinal nephritis will, unfortunately, sometimes pass into chronic Bright's disease, and then will require the usual treatment for this condition.

19 CATHEDRAL STREET.

BACTERIOLOGY.

BY W. WATSON CHEYNE, M.B., F.R.C.S.,
ASSISTANT SURGEON TO KING'S COLLEGE HOSPITAL, SURGEON TO THE PADDINGTON GREEN CHILDREN'S HOSPITAL, ETC.

THE rapid advances of bacteriology during the last few years, and the important *rôle* which bacteria play both in health and disease, render unnecessary any apology for the publication in a journal devoted to the medical sciences of a sketch of the chief facts which have been made out with regard to these microörganisms and of the methods of investigation employed in their study. A knowledge of what has been discovered with regard to these minute bodies is essential for the study of pathology, and for the rational treatment of infective diseases. But it is more especially in the department of preventive medicine that the practical value of these researches is as yet evident. So long as the precise cause of a disease is unknown, the views held as to its origin must necessarily be vague, and the measures adopted against it often either inefficient or excessive. But when the cause is known, and more especially when it can be studied apart from the body, its life history, its habitat outside the body, its mode of entrance into the system, and the best methods of destroying it under various conditions are learned, and the measures to be adopted against it can be made precise and effectual. It is not, however, sufficient for the medical officer of health to know the literature of the subject; he must himself be a bacteriologist, ready and able at any moment to carry out an investigation on bacteria. More especially must he be acquainted with the methods of demonstrating, recognizing, and studying these organisms in water, food, etc., with the view of determining in many cases whether these substances are hurtful or not, and also with the view of ascertaining the exact source and commencement of any given epidemic. The importance of this knowledge is self-evident, and I need not do more than point out one striking example of the value of being able to ascertain the earliest cases of an epidemic disease. The diagnosis, clinically or by post-mortem examination, of an isolated case of Asiatic cholera has, up till recently, been very difficult, or almost impossible; it is, as a rule, only after the epidemic is